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The Potential Virulence Factors of Providencia stuartii: Motility, Adherence, and Invasion

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Abstract

© 2018 Naziia Kurmasheva et al. Providencia stuartii is the most common Providencia species capable of causing human infections. Currently P. stuartii is involved in high incidence of urinary tract infections in catheterized patients. The ability of bacteria to swarm on semisolid (viscous) surfaces and adhere to and invade host cells determines the specificity of the disease pathogenesis and its therapy. In the present study we demonstrated morphological changes of P. stuartii NK cells during migration on the viscous medium and discussed adhesive and invasive properties utilizing the HeLa-M cell line as a host model. To visualize the interaction of P. stuartii NK bacterial cells with eukaryotic cells in vitro scanning electron and confocal microscopy were performed. We found that bacteria P. stuartii NK are able to adhere to and invade HeLa-M epithelial cells and these properties depend on the age of bacterial culture. Also, to invade the host cells the infectious dose of the bacteria is essential. The microphotographs indicate that after incubation of bacterial P. stuartii NK cells together with epithelial cells the bacterial cells both were adhered onto and invaded into the host cells.

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References

[1] S.-H. Wie, "Clinical significance of providencia bacteremia or bacteriuria," Korean Journal of Internal Medicine, vol. 30, no. 2, pp. 167-169, 2015.

[2] E. De Vecchi, S. Sitia, C. L. Romano, C. Ricci, R. Mattina, and L. Drago, "Aetiology and antibiotic resistance patterns of urinary tract infections in the elderly: a 6-month study," Journal of Medical Microbiology, vol. 62, pp. 859-863, 2013.

[3] D. Aubert, T. Naas, M.-F. Lartigue, and P. Nordmann, "Novel genetic structure associated with an extended-spectrum lactamase blaVEB gene in a Providencia stuartii clinical isolate from Algeria," Antimicrobial Agents and Chemotherapy, vol. 49, no. 8, pp. 3590-3592, 2005.

[4] M. Tumbarello, R. Citton, T. Spanu et al., "ESBL-producing multidrug-resistant Providencia stuartii infections in a university hospital," Journal of Antimicrobial Chemotherapy, vol. 53, no. 2, pp. 277-282, 2004.

[5] P. R. Krake and N. Tandon, "Infective endocarditis due to Providenda stuartii," Southern Medical Journal, vol. 97, no. 10, pp. 1022-1023, 2004.

[6] C. Simon, M. Dieli, A. Brucato et al., "Bacterial pericarditis due to providencia stuartii: An atypical case of relapsing pericarditis," Circulation, vol. 122, no. 4, pp. e401-e403, 2010.

[7] S. Unverdi, H. Akay,M.Ceri et al., "Peritonitis due to Providencia stuartii," Peritoneal Dialysis International, vol. 31, no. 2, pp. 216-217, 2011.

[8] O.R. Sipahi, S. Bardak-Ozcem, E.Ozgiray et al., "Meningitis due to Providencia stuartii," Journal of ClinicalMicrobiology, vol. 48, no. 12, pp. 4667-4668, 2010.

[9] J. Pizarro-Cerda and P. Cossart, "Bacterial adhesion and entry into host cells," Cell, vol. 124, no. 4, pp. 715-727, 2006.

[10] E. E. McClure, A. S. O. Chavez, D. K. Shaw et al., "Engineering of obligate intracellular bacteria: Progress, challenges and paradigms,"Nature ReviewsMicrobiology, vol. 15, no. 9, pp. 544-558, 2017.

[11] D. E. Johnson, C. V. Lockatell, M. Hall-Craigs, H. L. T. Mobley, and J.W.Warren, "Uropathogenicity in rats and mice of Providencia stuartii from long-term catheterized patients," The Journal of Urology, vol. 138, no. 3, pp. 632-635, 1987.

[12] S. Khashe, D. J. Scales, S. L. Abbott, and J. M. Janda, "Noninvasive Providencia alcalifaciens strains fail to attach to HEp-2 cells," Current Microbiology, vol. 43, no. 6, pp. 414-417, 2001.

[13] H. L. T. Mobley, G. R. Chippendale, J. H. Tenney, and J. W. Warren, "Adherence to uroepithelial cells of Providencia stuartii isolated from the catheterized urinary tract," Journal of General Microbiology, vol. 132, no. 10, pp. 2863-2872, 1986.

[14] C. Li, C. J. Louise, W. Shi, and J. Adler, "Adverse conditions which cause lack of flagella in Escherichia coli," Journal of Bacteriology, vol. 175, no. 8, pp. 2229-2235, 1993.

[15] J. Letourneau, C. Levesque, F. Berthiaume, M. Jacques, and M. Mourez, "In vitro assay of bacterial adhesion onto mammalian epithelial cells," Journal ofVisualized Experiments, no. 51, Article ID e2783, 2011.

[16] E. A. Elsinghorst, "Measurement of invasion by gentamicin resistance," Methods in Enzymology, vol. 236, no. C, pp. 405-420, 1994.

[17] C. E. Armbruster, S. N. Smith, A. Yep, and H. L. T. Mobley, "Increased incidence of urolithiasis and bacteremia during proteus mirabilis and providencia stuartii coinfection due to synergistic induction of urease activity,"TheJournal of Infectious Diseases, vol. 209, no. 10, pp. 1524-1532, 2014.

[18] E. C. Boyle and B. B. finlay, "Bacterial pathogenesis: Exploiting cellular adherence," Current Opinion in Cell Biology, vol. 15, no. 5, pp. 633-639, 2003.

[19] D. H. Stones and A. M. Krachler, "Against the tide: The role of bacterial Adhesion in host colonization," Biochemical Society Transactions, vol. 44, no. 6, pp. 1571-1580, 2016.

[20] G. M. Fraser and C. Hughes, "Swarming motility," Current Opinion in Microbiology, vol. 2, no. 6, pp. 630-635, 1999.

[21] P. N. Rather, "Swarmer cell differentiation in Proteus mirabilis," Environmental Microbiology, vol. 7, no. 8, pp. 1065-1073, 2005.

[22] N. Verstraeten, K. Braeken, B. Debkumari et al., "Living on a surface: swarming and biofilm formation," Trends in Microbiology, vol. 16, no. 10, pp. 496-506, 2008.

[23] R. M. Morgenstein, K. M. Clemmer, and P. N. Rather, "Loss of the waaL O-antigen ligase prevents surface activation of the flagellar gene cascade in Proteus mirabilis," Journal of Bacteriology, vol. 192, no. 12, pp. 3213-3221, 2010.

[24] L. Alberti and R. M. Harshey, "Differentiation of Serratia marcescens 274 into swimmer and swarmer cells," Journal of Bacteriology, vol. 172, no. 8, pp. 4322-4328, 1990.

[25] G. R. Nielubowicz and H. L. T. Mobley, "Host-pathogen interactions in urinary tract infection," Nature Reviews Urology, vol. 7, no. 8, pp. 430-441, 2010.

[26] B. B. Finlay and S. Falkow, "Common themes in microbial pathogenicity revisited," Microbiology and Molecular Biology Reviews, vol. 61, no. 2, pp. 136-169, 1997.

[27] P. Klemm and M. A. Schembri, "Bacterial adhesins: function and structure," International Journal of Medical Microbiology, vol. 290, no. 1, pp. 27-35, 2000.

[28] Y. Asahi, J. Miura, T. Tsuda et al., "Simple observation of Streptococcusmutans biofilm by scanning electron microscopy using ionic liquids," AMB Express, vol. 5, no. 1, 2015.

[29] T. Kwok, S. Backert, H. Schwarz, J. Berger, and T. Meyer, "Specific entry of Helicobacter pylori into cultured gastric epithelial cells via a zipper-like mechanism," Infection and Immunity, vol. 70, no. 4, pp. 2108-2120, 2002.

[30] J. E. Galan and D. Zhou, "Striking a balance: Modulation of the actin cytoskeleton by Salmonella," Proceedings of the National Acadamy of Sciences of the United States of America, vol. 97, no. 16, pp. 8754-8761, 2000.

[31] T. R. Garrett, M. Bhakoo, and Z. Zhang, "Bacterial adhesion and biofilms on surfaces," Progress in Natural Science, vol. 18, no. 9, pp. 1049-1056, 2008.

[32] P. Pletnev, I. Osterman, P. Sergiev, A. Bogdanov, and O. Dontsova, "Survival guide: Escherichia coli in the stationary phase," Acta Naturae, vol. 7, no. 4, pp. 22-33, 2015.

[33] S. L.Walker, J. A. Redman, and M. Elimelech, "Role of cell surface lipopolysaccharides in escherichia coli K12 adhesion and transport," Langmuir, vol. 20, no. 18, pp. 7736-7746, 2004.

[34] G. M. Bruinsma, M. Rustema-Abbing, H. C. Van Der Mei, and H. J. Busscher, "Effects of cell surface damage on surface properties and adhesion of Pseudomonas aeruginosa," Journal of Microbiological Methods, vol. 45, no. 2, pp. 95-101, 2001.

[35] J.Martinez,M.Mulvey, J. Schilling, J. Pinkner, and S. Hultgren, "Type 1 pilus-mediated bacterial invasion of bladder epithelial cells," The European Molecular Biology Organization Journal, vol. 19, no. 12, pp. 2803-2812, 2000.

[36] M. E. Konkel, D. R. Samuelson, T. P. Eucker, E. A. Shelden, and J. L. O'Loughlin, "Invasion of epithelial cells by Campylobacter jejuni is independent of caveolae," Cell Communication and Signaling, vol. 11, no. 1, p. 100, 2013.

[37] D. Ribet and P.Cossart, "Howbacterial pathogens colonize their hosts and invade deeper tissues," Microbes and Infection, vol. 17, no. 3, pp. 173-183, 2015.

[38] M. Bonazzi,M. Lecuit, and P. Cossart, "Listeria monocytogenes internalin and E-cadherin: From structure to pathogenesis," Cellular Microbiology, vol. 11, no. 5, pp. 693-702, 2009.

[39] S. L.Walker, J. E. Hill, J. A. Redman, and M. Elimelech, "Influence of growth phase on adhesion kinetics of Escherichia coli D21g," Applied and Environmental Microbiology, vol. 71, no. 6, pp. 3093-3099, 2005.

[40] A. M. Edwards and R. C. Massey, "Invasion of human cells by a bacterial pathogen," Journal of Visualized Experiments, no. 49, Article ID e2693, 2011.

[41] A.Mooney, C. Byrne,M. Clyne, K. Johnson-Henry, P. Sherman, and B. Bourke, "Invasion of human epithelial cells by Campylobacter upsaliensis," Cellular Microbiology, vol. 5, no. 11, pp. 835-847, 2003.

[42] C.H. Choi, J. S. Lee, Y. C. Lee, T. I. Park, and J. C. Lee, "Acinetobacter baumannii invades epithelial cells and outer membrane protein A mediates interactions with epithelial cells," BMC Microbiology, vol. 8, article no. 216, 2008.

[43] E. Eugene, I. Hoffmann, C. Pujol, P.-O. Couraud, S. Bourdoulous, andX.Nassif, "Microvilli-like structures are associated with the internalization of virulent capsulated Neisseriameningitidis into vascular endothelial cells," Journal of Cell Science, vol. 115, no. 6, pp. 1231-1241, 2002.

[44] V. Cano, D. Moranta, E. Llobet-Brossa, J. A. Bengoechea, and J. Garmendia, "Klebsiella pneumoniae triggers a cytotoxic effect on airway epithelial cells," BMC Microbiology, vol. 9, article no. 156, 2009.